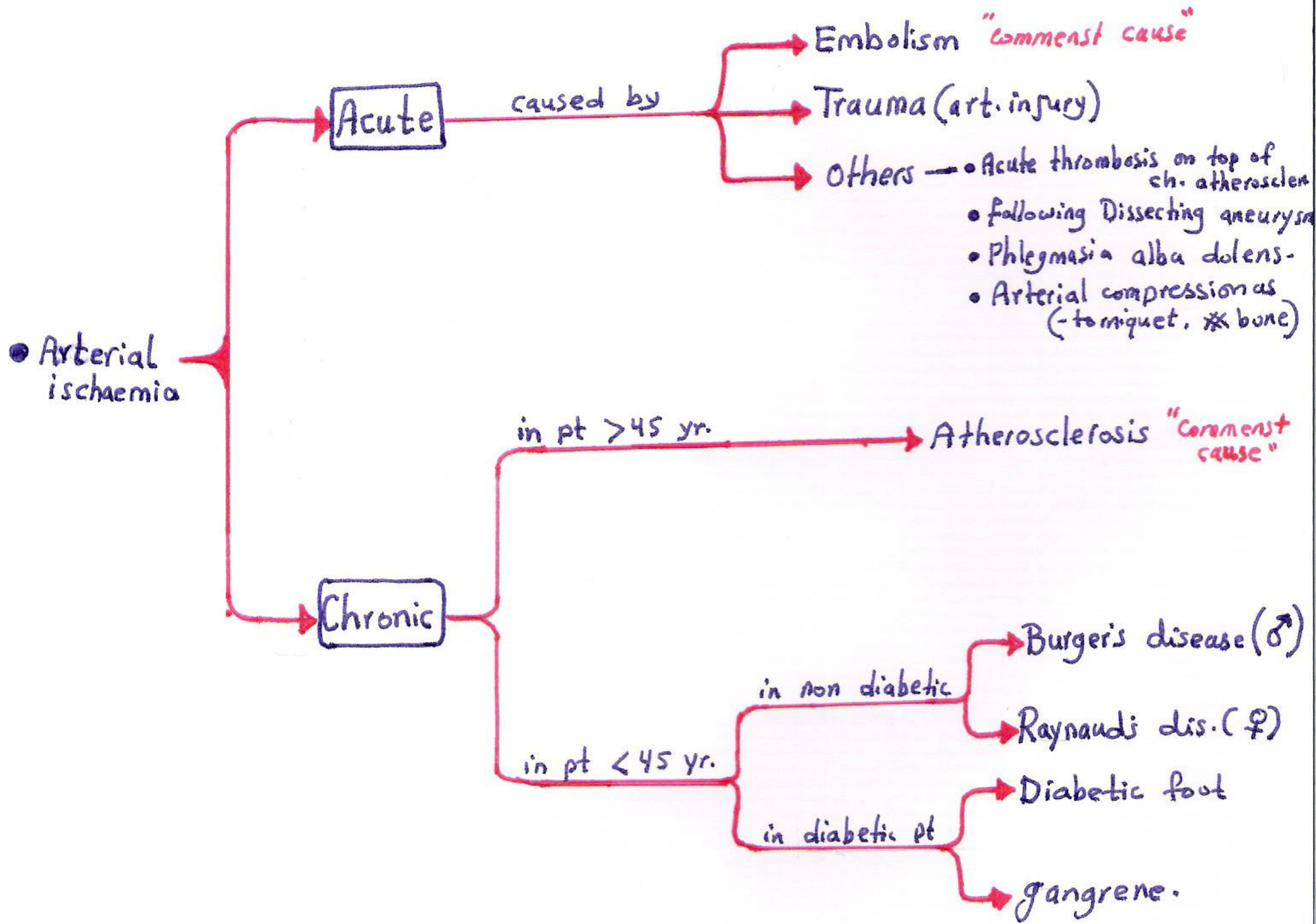
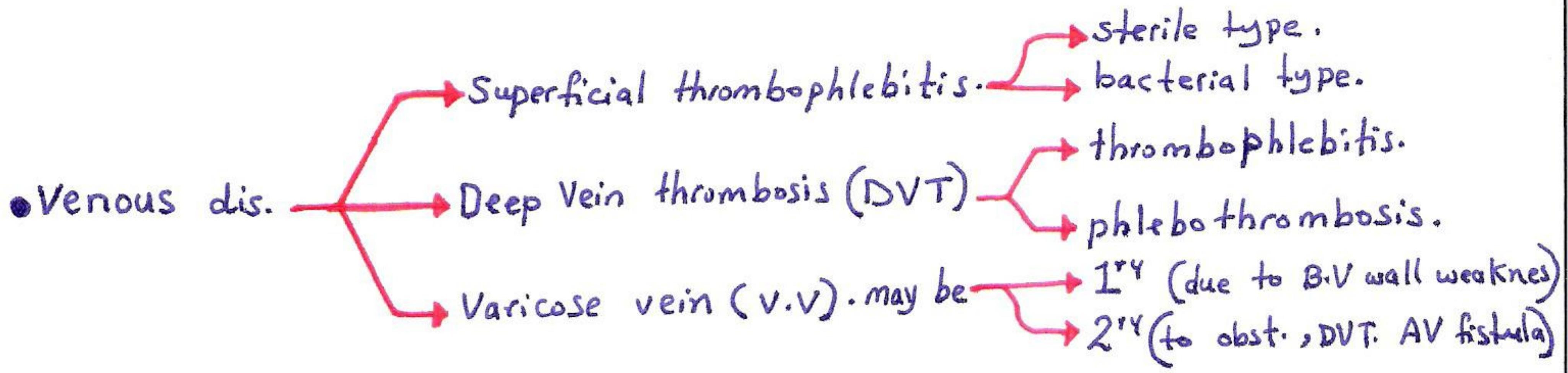


VASCULAR DISORDERS

The vascular disorders are mainly venous and arterial disorders (mainly arterial ischemia).



VISIT...

LANZAROTE
Caliente.COM

VENOUS DISORDERS

I - SUPERF. THROMOPHLEBITIS:

*Definition:- thrombosis of inflamed superficial veins.

*Aetiology:- ① systemic cause :- Polycythaemia, SLE, ulcerative colitis, Mondor's disease
② Local cause :- Varicose vein (v.v), Burger's dis., trauma iatrogenic (i.v infusion)

*Types:- ① Sterile type ② Bacterial type

*C/P :- -Symptoms: pain + FHMA (Fever, Headache, Malaise & Anorexia).
-Signs:- Tender Cord like structure ± overlying skin redness.

*Treatment:- ① Plastic bandage & underlying cause ② Analgesia & A.B (if bacterial type).

N.B.: thrombo-phlebitis Migrants [Trousseau sign] :- associated with internal carcinoma (as stomach & pancreas) due to increased viscosity of blood.

II - DEEP VEIN THROMBOSIS

*Definition:- thrombosis of deep veins, occurs more in calf veins

↳ valveless & sinusoids.

*Aetiology:- [Virchow's triad]:

① Vessel wall changes (Endothelial damage): due to trauma or inflammation.

② Blood flow changes (Venous stasis): in heart failure or compression as in pregnancy & tumour.

③ Blood composition changes (Hyper coagulability): due to:

- Oral contraceptive pills or polycythemia.
- deficient of - Antithrombin III or natural anti coagulant (as protein C & S).

*Risk factors → Other causes as old age & previous H/O DVT.
→ Commonly (>50%) after major operations [as *NOF, prostatectomy].
→ obesity & female on contraceptive pills.

*Types of DVT :-

The DVT may be due to Phlebotrombosis or thrombophlebitis.

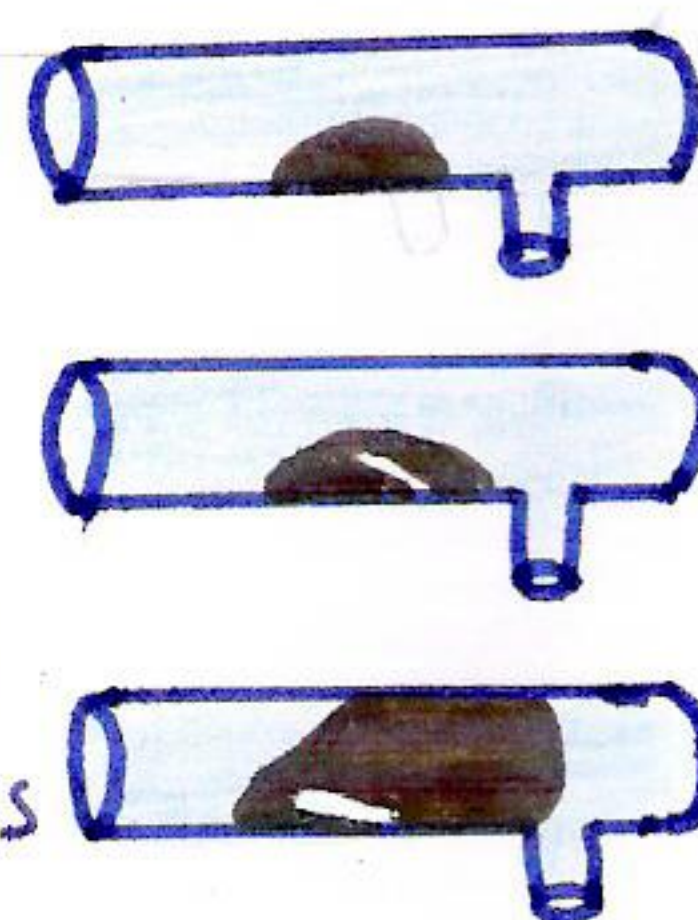
	Phlebo-thrombosis	thrombophlebitis
Definition	thrombosed <u>un</u> inflamed veins	thrombosed inflamed vein
Causes	Stasis or hyperviscosity	Draining inflamed organs
Site	commonly leg veins	commonly pelvic veins
Size of 1 st thrombus	Small	Large
Emboli	Common & Sterile	Rare & infective

*Pathogenesis:-

= Early stage: ①- Platelet adhere to endothelium of B.V forming "Gray cluster", mainly in calf veins.

②- fibrin & RBCs deposited in a layer between platelets forming "Line of Zahn".

③- After total occlusion of vein, thrombus spreads & the "Jelly-Like propagated thrombus" spreads as far as the near major tributary.



NB: At early stage pulmonary embolism is common because of loosely attached thrombus.

= Late stage: ④- the thrombus becomes tightly adherent → destruction of valves & occlusion of lumen = Post-phlebitic Limb. (see next)

⑤- by fibrinolysis → "Recanalization", (but valves destroyed permanently)

*Clinical picture [C/P]:

- The classic picture is ①-Pain :- aching discomfort commonly at calf. (25% are symptomatic)

②-Swelling:- ie oedema [reliable sign].

③-Tenderness:- on pressing muscle against bone.

= pt may be asymptomatic either silent or unexplained fever & ↑HR.

= pt may presents by pulmonary embolism as a 1st manifestation

= N.B - Symptoms & signs depends on the level of thrombosis

- Homan's sign (not used nowadays bec. may → embolisation) which is a +ve calf pain by sudden dorsiflexion.

NB:- the commonest vein for DVT is calf vein & rarest is IVC.

- The iliofemoral thrombosis may result in:

	① Phlegmasia alba dolens	② phlegmasia cerulea dolens
Definition	Painful white limb (Phlegma = inflammation)	- Painful blue limb.
Cause	Partial venous obst. (Less severe)	- Complete venous obst. (more severe)
C/P	Pallor: due to associated artery spasm	- Cyanosis (no venous return)
Complicat.	Coldness & ↓ pulsation	- venous moist gangrene.

* Investigation for DVT:

- ① Doppler U/S:- in completely obstructed leg vein shows absent flow & incompetent perforators "accuracy 85%"
- ② Coloured Duplex: shows an image of veins & bd flow direction "accuracy" 90-100%
- ③ Venography:- thrombus appears as a filling defect. [less used now].

* Complication of DVT:

- ① Early:
- ① Pulmonary embolism.
 - ② Venous gangrene (in phleg. cerulea dolens).

- ② Late:-
- ① Secondary V.V (varicose vein).

② Chronic venous insufficiency "post-phlebitis limb or syndrome"

- It is due to iliofemoral thrombosis with destroyed valves
 this → reflex of blood (through incompetent perforators) → high pressure in superficial veins especially during walking "Walking venous hypertension".

- In this case it • Non pitting oedema (fibrosis)

• Venous ulcer w may → Malignancy "Morgagni ulcer"
 → Periostitis
 → Talipes Equinov.

Blow-out syndrome

* Treatment of DVT:

- Prophylactic: General measures as adequate hydration, avoiding calf pressure & early post-op. mobilization.
- stop oral contraceptive pills (some school stop it 6 wk pre-op)
 othe school recommend heparin prophylaxis
- slight leg elevation (15-20°) after operation.

= prophylaxis for high risk patient include:

- Low dose heparin (mini-heparin) :- 5000 IU (s/c) : used 2 hr before operation & 12 hourly for 7 days or until mobilization.
- Low Molecular weight (LMW) heparin :- once daily.
 - it has lower risk of bleeding so more popular
- Calf compression devices intraoperative.
- graded compression stockings before operation until mobilization.

• **therapeutic ttt** : (anticoagulant).

- Giving heparin (i.v or s.c) or warfarin (oral), usually start by heparin then continue warfarin after.

	Heparin (i.v or s.c)	Warfarin
- Action	↑ activity of fibrinolysis	- Block synthesis of prothrombin & Factors VII, IX, X
- Dose monitoring	- APTT (must kept at 2.5 times the normal)	- PT (prothrombin time)
- Antidote	- Protamine sulphate	- Vit K.
- Complication	- Bleeding (over dose) - Failure of response (heparin resistance)	- Bleeding, peptic ulcer, - interaction with aspirin.
- Dose:	① i.v 5000 IU / 4 hourly. OR ② i.v 5000 IU plus then infusion of 5% glucose @ rate at 20-30 IU/Kg/hour. ③ s.c calciparin 150 IU/Kg/12 hr.	- Oral 10 mg initially then 5 mg for 5 days

NB :- Fibrinolytics (as streptokinase, urokinase & TPA) not used widely because of severe bleeding, allergy & very expensive

= Surgical Rx may be needed as venous thrombectomy in case of phlegmasia cerulea dolens by Fogarty catheter.

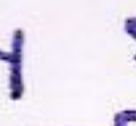
III - VARICOSE VEIN :

*Definition:- Dilated tortuous, elongated veins (superf. v. of lower limb).

*Anatomy:- The venous system of lower limb is either:

- ① superficial system → (Great & lesser saphenous veins)
- ② Deep system → (venae comitantes to arteries & venous sinuses inside calf ms "soleus").
- ③ Connecting system → connect superf. to deep.
 - Indirect communicators; ie from superf. → ms → deep veins
 - Direct comm. (perforators)
 - 1) three ankle perforators 2, 4 & 6 inches above medial malleolus (5, 10 & 15 cm)
 - 2) One perforator just below knee.
 - 3) one " at mid thigh.
 - 4) Sapheno-femoral junction
 - 5) Sapheno-popliteal → (in lesser saphenous)

*Types & causes of V.V :

	1 st V.V (non-obst.).	2 nd V.V (obstructive).
Causes :	- Congenital weakness of the bl. vessel wall → Dilated - vein → incompetent valves - Precipitated by long standing & sitting.	- obstruction of vein flow: e.g pregnancy, fibroid, ovarian cyst or any pelvic mass - Valve destruction as in DVT. - high flow & pressure as A.V fistula
C/P	- minimal oedema & skin changes & usually Bilateral, heaviness pain - The vein usually tubular  or sacular 	- Marked oedema & skin changes & usually Unilateral, bursting pain - The vein usually spider  or serpentine 
Past history	- No H/O DVT. - F/H of weak mesenchyme as • Varicocele • Piles • Visceroptosis • Hernia • Kyphosis • Flat foot	- H/O underlying cause as DVT

* Investigation of V.V. :-

- ① Venography :- shows patency & size of vein, valves & Blow out.
- ② Doppler u/s & Duplex → most imp.
- ③ Look for cause → abd. & pelvic u/s, Arteriography (A-V fistula)

* Complication of V.V. :-

- (A) Venous compl. →
 - 1) Hemorrhage.
 - 2) Superf. thrombophlebitis.
 - 3) Phlebolith (Calcified thrombosed vein).
- (B) Skin compl. →
 - 1) Brown discoloration → due to extravasated hemosiderins
 - 2) Dermatitis → due to irritant hemosiderin.
 - 3) Eczema → due to scratching dermatitis.
 - 4) Oedema → pitting, but later non pitting.
 - 5) Ulcer → Common after post-phlebotic cases.
 - 6) Malignancy → at ulcer site "Marjolin's ulcer."

* Treatment of V.V. :- "1st & cause if present"

① Conservative ttt :

- Indication :- early 1st V.V, pregnant, pt waiting, unfit or refusing surgery
- Methods :- below knee stocking, elevation of leg, regular exercise & avoid long standing & sitting.

② Injection-compression sclerotherapy :

- Aims to occlude the vein by fibrosis
- Indication :- 2nd V.V (spider type) & residual veins after operation.
- Contraindication :- 2nd V.V & DVT, pregnancy & acute ^{septic} thrombophlebitis
- Method :- inject the vein after emptying & apply firm elastic bandage for 2 wk (some schools for 6 wk).
 - Using either ① 3% Na tetradecyle sulphate.
 - ② 5% Ethanolamine oleate. or
 - ③ 5% Na Morrhuate.
- Use a small dose (1 ml) and inject only one vein & others at other visits & advice exercise.

③ operations of V.V. :-

- Indication :- 1) Saphena varix & Blow out.
2) repeated superf. thrombophlebitis.
- Subcutaneous stripping of long saphenous is the common operation, done by ligating long saphenous & its tributaries then stripping of whole long saphenous.
- NB : Ligation of vein & out stripping is called Trendelenberg's operation done in case of sapheno femoral incompetence.

NB : Saphena varix : [if 1st V.V.] : is a saccular dilatation shows expansile impulse on cough at sapheno-femoral junction.

* Examination of pt. in V.V. :-

- ① General Exam :- look for cause (in 2nd V.V) and look for signs of weak mesenchyme (in 1st V.V).
- ② Local Exam :- pt must be standing & exposed.
 - A Inspection :- for side (uni- or Bilateral), site (below or above knee) shape (tubular, spider, ...), skin over (as pigment, ulcer.) swelling (oedema) and skeletal changes (as Talipes equinus)
 - B Palpation :- V.V are soft compressible, but may found tender cords (thrombophlebitis), thrill (A-V fistula)
 - C Percussion :- "Schwartz percussion" : percussion of vein proximally by index finger & relieving by another index distally if the wave transmitted distally → ie incompetent valve.
 - D Auscultation :- in A-V fistula → continuous machinery murmur.
 - E Special test :-
 - To test blow out (incompetent perforator) →
 - ① Trendelenberg test.
 - ② Multiple Tournique test.
 - ③ Manual localization test
 - ④ Fegan's test.
 - To test deep veins (patent or closed) →
 - ① Perthe's test (not done)
 - ② Modified Perthe's test.

* Special tests for V.V :

(A) To detect blow out = "incompetent perforators"

① Trendelenberg test:-

- pt lies down, empty the vein (massage) & apply tourniquet just below saphenous opening & ask pt to stand up
- Result
 - slow filling from below → Normal
 - Rapid filling → blow out "incompetent perforators"
 - No filling, so remove tourniquet → Rapid filling from above → ie sapheno-femoral incompetent.

② Multiple tourniquet test:-

- pt lies down, empty the vein, & apply ³ tourniquet one just below saphenous opening, other above knee & one below knee & ask pt to stand up
- Result
 - Rapid filling of any segment means blow out in that segment removed.



③ Manual localization test "two fingers test"

- pt stand & press by index at the great saph. vein & by other index empty the vein in opposite direction
- Result: if rapid filling between your index fingers → blow out



④ Fegan's test:-

- pt stand & mark the varicose veins & lie the pt down & detect the defect of deep fascia "blow out"

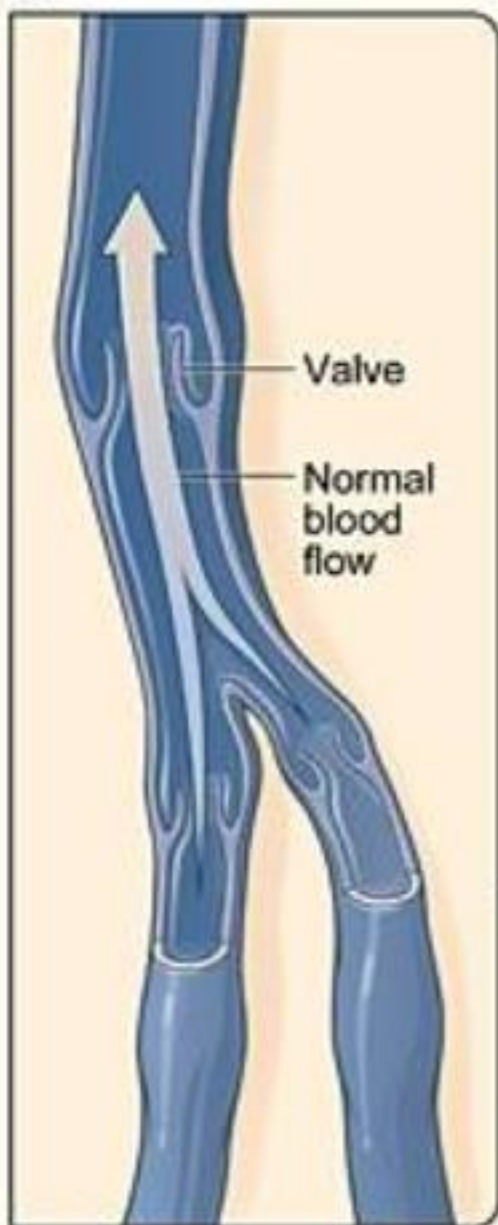
⑤ To test the deep veins if patent:-

① Perthe's test:- (not done): it depends on pain.

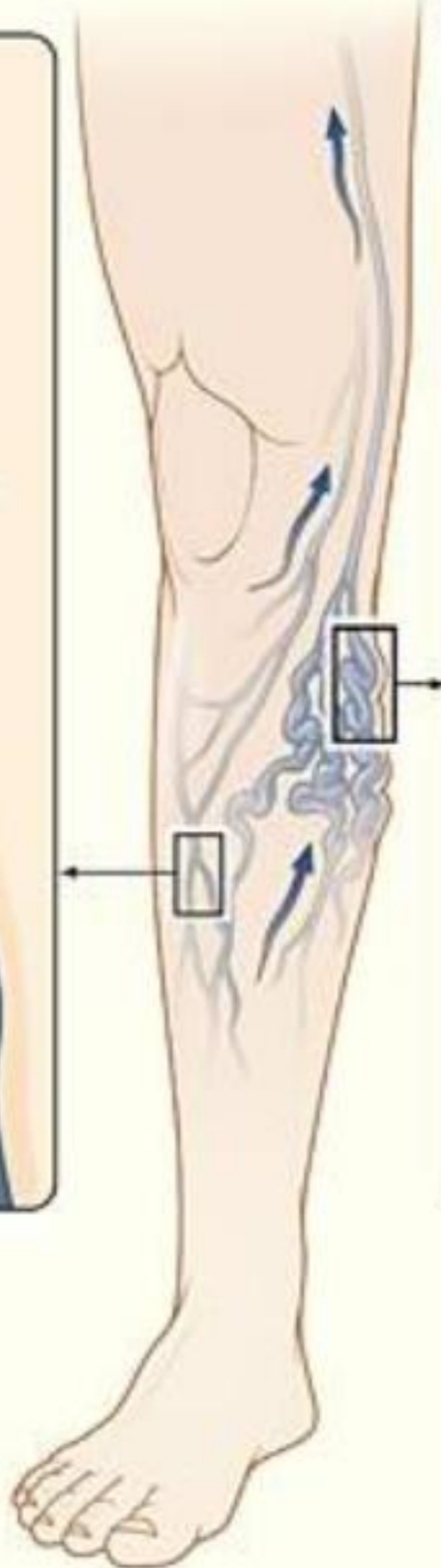
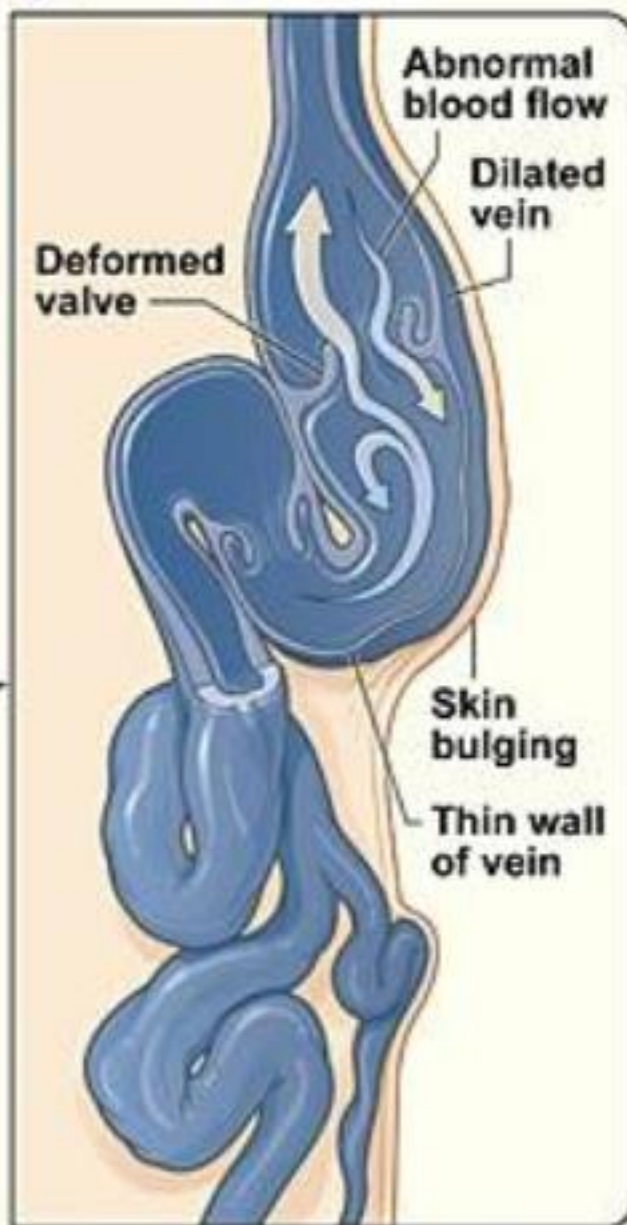
② Modified perthe's (Ochsner's test):-

- pt stand & tourniquet applied below saphenous opening & ask pt to walk insitu for 10 min
- Result:
 - if engorge (prominent) veins → occluded deep system
 - if shrunken veins → patent deep system.

A Normal vein



B Varicose vein



"Chronic Leg ulcers"

* **Definition:-** Ulcers is a discontinuity of the skin.

* **Classification:-** ① Congenital:- e.g. spherocytosis & sickle cell anemia.

② Traumatic:- Trauma & bed sores

③ Inflammatory: T.B & Φ ulcers.

④ Nervous:- Neurotrophic ulcer (e.g. Φ neuritis)

⑤ Lymphatic:- Lymphoedema ulcer.

⑥ Arterial:- ischemic ulcer.

⑦ Venous:- venous ulcer.

⑧ Neoplastic:- Marjolin ulcer.

* **C/P:- inspection:-** - Number of ulcers, site, size, shape.
- Margin, edge, floor & discharge

- **Palpation:-** - Temp., tenderness, base (soft or hard, fixed or not)

* **How to differentiate:**

• Venous ulcer $\rightarrow$ H/o varicosities, sloping or punched out edge, brown discolor usually at medial side above med. malleolus ↓ pain by foot elevation

• Arterial ulcer $\rightarrow$ H/o claudication, ↓ pain by foot dependency with deep ulcer, usually in foot.

• Neoplastic $\rightarrow$ Raised everted edge, hard & fixed base & hard LN

• inflammatory $\rightarrow$ Undermined edge  in TB & punched out in Φ 
usually at metaphysis of Tibia in TB & middle 2/4 of tibia in Φ with caseous material in TB

• Traumatic $\rightarrow$ Punched out edge, usually at middle 2/4 of tibia

• Congenital $\rightarrow$ usually at middle 2/4 of tibia.

• Neurotrophic $\rightarrow$ usually at sole of foot with callosities around

• Lymphatic $\rightarrow$ usually at dorsum of foot & associated with swollen limb & lymphorrhea.

[NB] sloping ulcer  usually indicate healing (granulation).

• Bed sores (Decubitus ulcer) usually at pressure sites as heels, sacrum, ischial tuberosity, greater trochanter & scapular blades

$\rightarrow$ the 10/5

ARTERIAL DISORDERS

- The arterial disorders may be
 - ▀ Ischaemia (acute & chronic)
 - ▀ Aneurysm.
 - ▀ Gangrene
 - ▀ Arteriovenous fistula.

ACUTE ISCHAEMIA

*** Definition:** - Ischaemia:- is a ↓ed arterial supply sufficient to interfere w nutrition & function of the limb.
- Acute ischaemia is a sudden total occlusion of artery.

NB:- Acute ischaemia is more serious than chronic because No time for development of collaterals.

- * Aetiology:**
- ① Embolism "the commonest cause".
 - ② Trauma (arterial injury) "incidence ↑ing nowadays".
 - ③ Others: e.g - Phlegmasia Alba dolens.
 - Arterial compression (bone, tourniquet, Ligature, ...)
 - Complicating dissecting aneurysm.
 - Acute thrombosis on top of atherosclerosis.

*** Pathophysiology:** - Sudden artery occlusion → Reflex spasm of nearby vein which → tissues loaded w blood → gangrene (moist aseptic)
- Muscle irreversible damage occurs within 6 hrs while that of skin within 24 hrs.

- * Clinical picture:**
- ① **Pain:** - sudden, bursting or cramp like ↑ by movem. or warmth.
 - ② **Pallor:** - 1st pale then mottled cyanosis & may → gangrene
 - ③ **Parasthesia:** - due to nerve ischaemia (1st ^{skin} hyposthesia then anaesthesia)
 - ④ **Paralysis:** - e.g paraplegia in aortic bifurcation occlusion.
 - ⑤ **Pulselessness:**
 - ⑥ **Progressive coldness.**

Embolitic Ischemia

Definition

- Sudden impaction of an embolus in a narrow blood vessels.

Incidence

- Commonest esp. in Cardiac pt.

Aetiology

- Embolus usually from
 - Lt atrium → MS & AF
 - Lt ventricle → recent MI.
 - Valves → subacute bact endocarditis
 - Paradoxical embolus → in ASD, VSD
 - Aorta → aneurysm.
 - Atheromatous plaques → atheroma
 - Rarely → fat, air, clot, Tumor cells
- Site of impaction is vessel bifurcation due to
 - ↓ diameter
 - slowing of bd. flow
 - Turbulence of flow

C/P

= 6 Ps ± signs of heart problem

Investigation

- Detect cause → ECG, Echo, ...
- Detect art. obst → Angiography.
 - Doppler uls
 - Duplex scanning

Treatment

- ① Urgent Embolectomy :- done under local by Fogarty balloon catheter & give preop. heparin (prevent thrombus propagation) & postoperative.
 - it must be within 6 hrs
 - if after called delayed embolectomy where it only lowers level of amputation
- ② Amputation :- in established gangrene
- ③ Fibrinolytics :- ?? trial (not effective)

Complication of Embolectomy : are

- ① Sudden death by pulm. embolism.
- ② Compartment syndrome → do fasciotomy.
- ③ Reperfusion injury as ↑ K⁺ & Myoglobin from dead ms → cardiac arrest (K) or renal failure (myoglobin)

Traumatic Ischemia

- Sudden interruption of artery supply of a limb by injury

- Common nowadays (RTA, war...)

- ① Open injury → stab, bullet, ...
- ② closed " → * as subcondyl * of Femur or humerus
- Complete division of artery 
 - bleeds profusely but stops or ↓ soon as V.C with contract. of intima & media (no puls distal)
- Partial division : not stopped bec of contraction & retraction of arterial wall, Puls is weak.
- Injury without bleeding occurs in ① art. spasm as in irritation by missile around. 
- ② Art. contusion ± thrombus 

= 6 Ps + H/o trauma.

- If shocked pt → resusc then immediate surgical exploration
- Cold cases → X-ray (bullet...) angio, Doppler, Duplex.

- ① If shocked pt → resusc & ABC assessment
- ② Control bleeding by compression or surgical
- ③ Immediate exploration.
- ④ Fasciotomy (to prevent the compartment syndrome)
- ⑤ In arterial spasm & contusion excision & venous graft done
 - some try spasmolytic (e.g papaverine) & heparin in spasm
- ⑥ Rx of * bone, antibiotics, anti tetanus, ...

CHRONIC ISCHAEMIA

* **Definition** :- Slowly progressive arterial obstruction.

* **Aetiology** :-

- ① Atherosclerosis "commonest cause."
- ② Arteritis : eg Burger's disease.
- ③ Vasospastic dis. eg Raynaud's disease
- ④ Diabetic foot & gangrene.

* **ATHEROSCLEROSIS** :

* **Definition** :- Degenerative arterial disease due to Aging process affecting mainly Large & medium sized arteries.

* **Incidence** :- old (>40 yr) (male > female)
- Risk factors : heavy smokers, hypercholesterolemia, hypertriglycerides, hypertension, obesity & +ve family history.

* **Pathology** :- the 1st pathology called "Atheroma" which is elevated yellow patches at intima & subintimal ↑ in Lipid & CT and at media & adventitia & fibrosis.

* **C/P** :-

- ① **Pain** :- intermittent claudication or rest pain.

② **Pulse changes** :- artery palpated (thick), ↓ amplitude or absent.

③ **Progressive coldness** :- may be False warm as ① covered ② infected ③ sympathectomy ④ DM "autosympathectomy."

④ **Cutaneous changes** :- hair loss : Nail → brittle ; skin → dry, scaly with ulceration, infection at pressure sites.
- paraesthesia (gradual loss), tingling & numbness.

⑤ **Colour changes** :- the skin colour of lower limb may be:

- Normal colour → mild ischemia
- Fixed change → severe ischemia (pregangrene) gradually
- Postural change → Do "Burger's test" → elevate limb -
 - normally no change on elevation
 - Pallor at elevation
 - cyanosis at lowering } ischemia

Burger's angle = angle at which limb becomes pale on elevation from horizon
• smaller the angle, advanced ischemia

* Investigation of chr. ischemia:

- 1] Clinical :- to detect degree of pain [claudication time, distance & rest time]
- to detect colour change - [Burger's test & angle]
 - capillary filling test
 - venous filling test} see later
- 2] Laboratory :- Urine (for sugar), blood (CBC - anemia, FBS - DM, Lipid profile)
- 3] Radiological & instrumental
 - Arteriography (the main) - to show

{

condition of artery

site & length of obst

collateral condition

Distal run off (distal flow after obst.)

 - Done by Seldinger's needle.
 - Doppler u/s :- to show : bd flow, degree of ischemia & site of obst
 - to measure "Ankle-Brachial pressure index" which is a ratio bet. pressure of ant. tibial a & brachial a normally ≥ 1 →
 - if < 0.9 → mild ischemia to moderate
 - if < 0.7 → Severe ischemia
 - if < 0.3 → Impending gangrene
 - Coloured Duplex : (most imp. invest) → imaging & blood flow measure
 - ECG & stress ECG → for coronary & myocardial condition.

* Treatment of chr. ischemia:

- ① Conservative :- if mild ischemia or unfit pt.
 - relief symptoms by improving general health (anemia, heart lesion)
 - relief pain & careful washing & drying of ischemic limb
 - improve bd. supply by - VD drug as Torental or PGE₁
 - antiplatelet as aspirin or Persantine.
 - stop smoking, regular exercise, control of DM & HbT.
- ② Endovascular surgery: for localized obst. ^{< 10 cm} in large or medium a but may restenosis, done either by.
 - A] Percutaneous transluminal balloon angioplasty (PTA) :-
balloon catheter to dilate stenosed part
 - B] Arterial stents :- after PTA to keep lumen patent
 - C] LASER angioplasty :- destruction of atheromatous plaques.

③ Surgical options:- [Indicated with rest pain + Run off]

① Thrombo-endo-arterectomy:-

- for localized lesion in large artery.
- Arteriotomy done & removing thrombus & thickened wall (intima) & after closure patent lumen will epithelize

② Arterial by-pass:-

- for multiple lesion in large medium arteries
- e.g - Aorto-iliac & Femoro-popliteal bypass [anatomical]
- Axillofemoral & femoro-femoral bypass [extraanatomical]
- [NB]** extraanatomical done in unfit pt as in COPD, ... the graft used is natural (saphenous) in medium a or synthetic (eg Teflon or Dacron) in large a

③ Profundo plasty:- ie dilatation of profunda in case of super. femoral block.

④ Lumbar sympathectomy:- [Rest pain + No Run off] or after amputation to improve wound healing.

- done either surgical (L2.3 ganglia) in fit pt or chemical by xylocain 1% (temporary) or phenol 5% (permanent)

⑤ Amputation:- either

- Conservative amputation: at line of demarcation.
- Urgent high amputation: if spreading gangrene, Toxemia done by Above or below knee amputation.

* ARTERITIS:

The arteritis may be

- Burger's disease.
- infective arteritis.
- Takayasu's arteritis (autoimmune, young ♀)
- Polyarteritis nodosa (collagen dis. ass. w SLE, scleroderma)
- End arteritis obliterans (\$, rarely TB) → rare now bec. of good ttt.

* BURGER'S DISEASE:

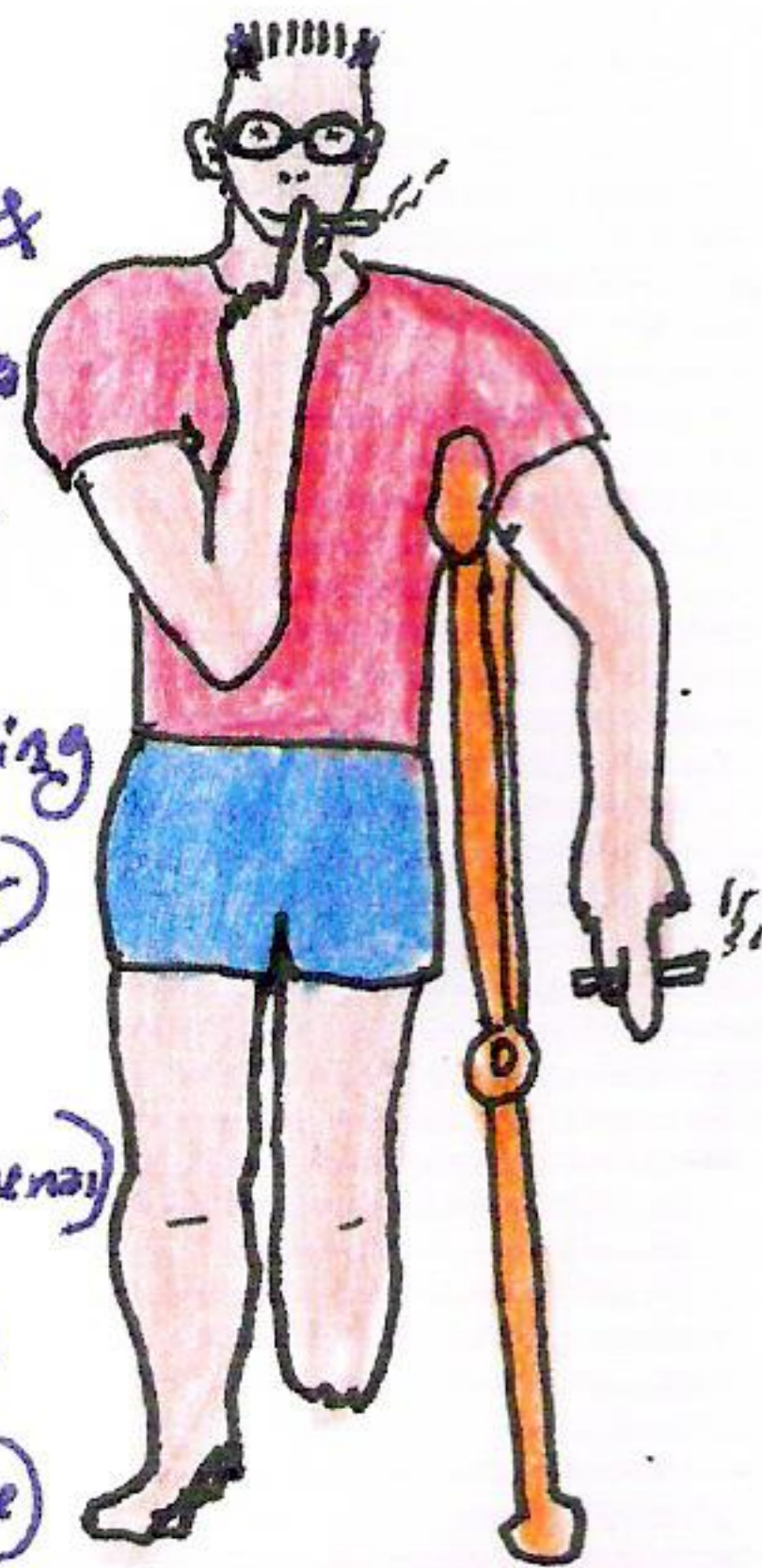
"Thrombo-Angitis Obliterans"

* Definition: Inflammation & thrombosis of small arteries & veins & perivascular fibrosis (blending vessel & nerve into one mass → early neuritis & severe rest pain).

* Aetiology: unknown, may be started by spasm & then thrombosis → fibrosis, spasm is due to smoking

* C/P → it is disease of heavy smokers, ♂, young (<40yr) involving upper & lower limbs with early rest pain & limited gangrene (+ Raynaud's phenomenon)
foot claudication, sup. thrombophlebitis

* Treatment: - stop smoking & sympathectomy → main ttt
- may conservative amputation (bypass no value)



NB: Burger's dis. differ from atherosclerosis where atherosclerosis is dis. of old age (>40yr) more in male (but burgers 95% are ♂) & no upper limb ischemia, late rest pain, claudication & the ttt. is mainly bypass.

* VASOSPASTIC DISORDERS:

RAYNAUD'S DISEASE:

* Definition: vasospastic dis. affecting digital arteries, ^{bilateral} young ♀, in cold

* Aetiology: Unknown:- but may be cold oversensitivity (not sympathetic oversensitivity as sympathectomy give early relief then recure).

* C/P: → • Pallor (due to spasm) & dull pain then
• Cyanosis (spasm → ↑ metabolites → congestion), burning pain then
• Rubor (relax. of artery & attach ends) → pain disappear.

→ pt is normal between attacks.

• Late stages → trophic changes (Hair loss, Nail brittle, dry scaly skin)
superf. ulcers & dry gangrene in finger tips.

* Management in Raynaud's dis. (success in early stages)

- Conservative treatment [avoid cold + Trental = vasospastic drug].
- Sympathectomy: in severe cases but after months mild sensitivity to cold returns.

NB: Raynaud's phenomenon :- is of known cause ie 2^{ry} to

- Systemic dis. → Collagen dis (scleroderma), myxoedema, ...
- Compression syndrome → e.g. carpal tunnel, thoracic outlet syndrome
- Occupational → vibration (drill, chainsaw), piano playing, typing

* **GANGRENE** :

* Definition:- Death & putrefaction of a Macroscopic tissue in a living person

- * Aetiology:-
- Arterial obst. (acute or chr.).
 - Venous gangrene (phlegmasia cerulea dolens).
 - Neuropathic " (Leprosy).
 - Infected " (I^d gangrene, Gas gangrene).
 - Traumatic " (injury, Bed sores).
 - Physiochemical " (Burns & Frost bite).

NB:- Atherosclerotic (arterial) & infected are the most common

- * Types:-
- Dry gangrene: with chronic ischaemia
 - Shrunken, wrinkled, Hard limb, no odour
 - Moist gangren: with acute isch. (chr. isch. on top of oedema as HF)
 - Swollen, oedema, toxemia, offensive odour
 - Can be infected → very rapid spread gang
- Usually dry gangrene has line of demarcation & line of separation with band of hyperemia & anaesthesia.

* Treatment:- Amputation; either

- Conservative amputation: if good adjacent bd. supply with line of demarcation & separ. clear
- Urgent high amputation:- in spreading gangrene, toxemia either - Above knee amb. (AKA) or below knee (BKA)

* ANEURYSM :

* Definition: arterial aneurysm is a sac filled by bd. communicating with lumen of an artery.

* Classification: classified according to:-

- I- structure**: ①- True aneurysm:- wall of aneurysm is stretched wall of the artery (ie formed of 3 layers)
②- False aneurysm:- wall formed of fibrous tissue due to organization of pulsating hematoma.

II- shape: ① Fusiform. ②- saccular ③- Dissecting.

III- Aetiology: ①- Congenital:- e.g Berry's aneurysm (in circle of Willis)

②- Acquired:- **A**- pathological: commonest causes:

- Atherosclerotic (commonest) subacute inf end
- Mycotic aneurysm → infected emboli in SIE
- Collagen dis → Marfan's synd., Ehler's dis

B- Traumatic → weak wall → True aneurysm
→ pulsating hematoma → False "

* Clinical picture: - Usually asymptomatic, or s/s of underlying cause
o/e:- pulsating round smooth mass (↑ by distal pressure & ↓ by proximal pressure)
moved side to side (but not along course of the artery)

* Complication: ① rupture → shock.

②- infection.

③- Thrombosis & Embolism.

④- Distal ischemia

⑤- Compression symptoms

Vein → oedema & varicosities.

Artery → ischemia

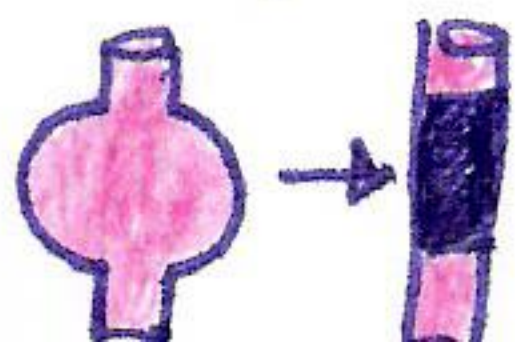
Nerve → sensory changes

Bone → erosion of bone.

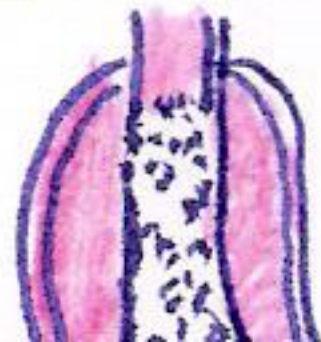
* Investigation: - Plain X-ray (calcifi-), arteriography (useless if clotted aneurysm)
- Sonar & CT scan (the choice) & Duplex scanning.

* Treatment: - If asymptomatic & < 5cm → follow up & sonar/6 months
- if > 5cm → surgical graft either

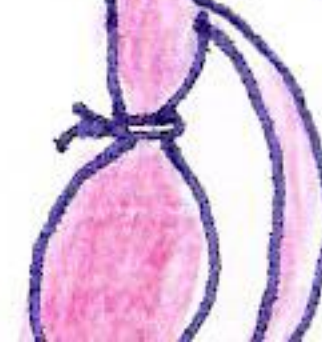
Excision + Graft
(classical)



Graft inside aneurysm
(for > 5cm)



Excision & by pass graft
(if surrounding imp. structure)



Abdominal Aortic Aneurysm: "AAA"

- The AAA is the commonest arterial aneurysm,
- It affects male > Female, age 50-60 yrs (atherosclerosis)

* Clinical picture: 75% are asymptomatic & may be discovered accidentally as pt may complain of vague abd. pain or chronic back pain [due to bone compression & interfering with vertebral blood supply] misdiagnosed as disc prolapse.
- pt may be presented by symptoms of rupture: i.e. acute (sudden) upper abd. pain at back or flanks and s/s of shock.

* invest. , treatment → see aneurysm.

* ARTERIO-VEINOUS FISTULA:

* Definition: Abnormal connection between an artery & vein.

* Types & aetiology: - A-V fistula may be:

- ① Congenital: - usually small & multiple, more in lower limb → Giant limb.
- ② Acquired: - Trauma (stab, bullet...) or iatrogenic (hemodialysis, arteriovenous)

* Clinical picture: pain & swelling in area of fistula with enlarged tortuous artery & veins, ↑ed skin temp., venous hypertension & ischemia distally, cont. machinery murmur ^{bruit}

* investigation: MRI angio, Duplex & Doppler.

- angiography done in case of decision to do embolization

* Treatment: - Excision & vascular repair done in large fistula
- for small one either follow up (if complicated → surg)
- Some advice embolization of fistula.

* Complication: cosmetic, Hge, thrombosis, distal ischemia, venous hypertension, high output cardiac failure

Branham's sign: it indicates slowing of heart rate as soon as fistula is compressed.

* DIABETIC FOOT :

* **Definition:** It is a serious foot infection ± gangrene in diabetic pt.

* **Incidence:** 20% of D pt will have D foot & it is the most common cause of LL amputation. [20% of D ulcers ends by amput.].

* predisposing factors:

- ① **Vascular** :- Macroangiopathy → by atherosclerosis mainly leg vessels.
- Microangiopathy → by arteritis

NB: atherosclerosis differs from non diabetics in that
1- appears early & affect vessels below knee.
2- more progressive as it is continuous lesion (not patchy)
3- abnormality mainly in basement membr. of endothelium

- ② **Neuropathy** :- DM affects both sensory & motor systems :-

⚙ **Sensory** :- Sensory loss takes "Glove & Stocking distribution"
[It is due to myelin deficient synthesis] → susceptible to trauma (unware) → ulceration & infection

NB: Sensory loss is for deep sensation (vibration & joint position) before superf (pain, touch & temp.)

⚙ **Autonomic** :- (auto-sympathectomy) :

⇒ Loss of ms tone → vein dilation → congestion & oedem → skin stretching → skin breakdown (ulcer) → infection.

⇒ Anhidrosis: Less sweating → dry skin → ↑ infection.

⚙ **Motor** :- mainly small ms. of foot → imbalance → deformity → ↑ trauma.

- ③ **Immunodeficiency** :- it is represented by:

⇒ ↓ glycosylated Ab formation.

⇒ ↓ phagocytic activity of Leucocytes.

⇒ ↓ delivery of Ab & phagocytes to site of infection (microangiopathy)

* Investigation :-

- CBE (Hb, TLC) - FBS, HgA_{1c} (glycosylated Hb), urine for sugar
- X-ray → gas under skin, bone change (osteomyelitis)
- Culture & sensitivity (from discharge or blood).
- Vascular assess (Doppler, arteriogram, ...)

* Clinical picture:

- H/o minor trauma, with foot infection, offensive odour & gangrene or H/o complication as osteomyelitis.
- examine locally (see ulcer exam. 100) and generally (DM is multi-systemic).

* Management:

① prevention & education:- control DM, good hygien, avoid trauma.

② Medical ttt:- it includes:

a- control of DM:- as infection ↑ level of glucose & vice versa ie makes difficult control so change into insulin if pt on oral hypoglycemic.

b- control of infection:- done by:-

- Debridement of necrotic tissue ^{mechanical or chemical (H₂O₂)} if after it is bleeding & oozing (ie neuropathy mainly) if no oozing & bleeding (ie vascular-ischemia) → "angiography"
- Dressing (always moist). & pus drain.
- systemic antibiotics (broad spectrum) see c/s.
- NB in chr. osteomyelitis debridement is mandatory

③ Surgery:- amputation for gangrene

④ Correct underlying diseases as anemia (to ↑ tissue O₂) and cardiac dis., Tri B complex for sensory loss.

* Stages for D.F infection:

① stage of cellulitis: skin erythema by streptococcal infection or staph (in staph is bullae formation). No Pus. No discharge

② stage of Deep skin & soft tissue infection: pt has severe pain
NB:- the D/D is compartment syndrome or gas gangrene

③ stage of acute osteomyelitis: pt has pain in bone + foul discharge

④ stage of chronic osteomyelitis: pt has continuous discharging sinus with s/s of chr. osteomyelitis

⑤ stage of fetid foot: severe inflam. of soft tissue & bone. pt has a foul smelling.

* Stages of D.F ulcer :-

- Stage 0 - preulcerative or post ulcer $\bar{e}$ fully epithelized.
 - A $\rightarrow$ pre-or post-ulcer $\bar{e}$ out cellulitis.
 - B $\rightarrow$ " " " " $\bar{e}$ cellulitis.
 - C $\rightarrow$ as A $\bar{e}$ signs of regional ischemia.
 - D $\rightarrow$ " " " " " severe ischemia.
- Stage I - Ulceration (partial or full thickness) $\bar{e}$ out deep structure
 - A $\rightarrow$ deep ulcer $\bar{e}$ out cellulitis.
 - B $\rightarrow$ " " $\bar{e}$ cellulitis.
 - C $\rightarrow$ " " $\bar{e}$ regional ischemia.
 - D $\rightarrow$ " " $\bar{e}$ severe ischaemia.
- Stage II - Ulceration $\bar{e}$ soft tissue infection $\bar{e}$ out joint involve.
 - A $\rightarrow$ $\bar{e}$ out cellulitis. C $\rightarrow$ regional ischemia
 - B $\rightarrow$ $\bar{e}$ cellulitis D $\rightarrow$ sever ischemia.
- Stage III - ulceration $\bar{e}$ joint & bone involvement (osteomyelitis)
 - A $\rightarrow$ $\bar{e}$ out cellulitis C $\rightarrow$ regional ischemia.
 - B $\rightarrow$ $\bar{e}$ cellulitis D $\rightarrow$ Sever ischemia.
- Stage IV - fore foot gangrene (toes).
- Stage V - Whole foot gangrene.

• Fontain classification: for chronic ischaemia :

- Stage I $\rightarrow$ asymptomatic
- Stage II $\rightarrow$ intermittent claudication
- Stage III $\rightarrow$ rest pain
- Stage IV $\rightarrow$ gangrene

• Boyd's classif. : for degree of ischemia (claudication):

- Stage I $\rightarrow$ $\uparrow\uparrow\uparrow\uparrow$. Pain (claudi) starts on walking & $\downarrow$ by continue walking (good collateral) $\rightarrow$ mild ischemia
- Stage II $\rightarrow$ $\uparrow\uparrow\uparrow$. Pain starts on walking & gradually $\uparrow$ by walking ie $\rightarrow$ moderate ischemia.
- Stage III $\rightarrow$ $\uparrow$ - Pain starts on walking but fore the patient to stop $\rightarrow$ Sever ischemia

LYMPHATIC DISORDER

LYMPHOEDEMA

* Definition :- hypertrophy of skin & s.c tissue by chronic lymphatic obstruction.

* Causes & types :

① Congenital (1st) - [rare] manifested either $\left\{ \begin{array}{l} \text{at birth} \rightarrow \text{Milroy's dis.} \\ \text{at puberty} \rightarrow \text{Precox.} \\ \text{at adult} \rightarrow \text{Tarda.} \end{array} \right.$

② Acquired (2nd) - [common] -

- Traumatic : e.g Burns, LN block dissection.
- inflammatory : e.g Filariasis, TB lymphangitis, ..
- Neoplastic : e.g Metastasis, Lymphoma.

* Stages of lymphoedema :-

- Stage I :- soft pitting oedema.
- Stage II :- Lymphorrhoea due to rupture of lymph vessels.
- Stage III :- Non pitting oedema due to Fibrosis of skin & s/c tissue
- Stage IV :- Warty-pseudo-papillomatous formation :- ie elephantiasis.

* Complication :-

- Recurrent cellulitis & Lymphangitis (commonst).
- Lymphoedema ulcer & infected blebs.
- heavy & huge limb.
- Lymphangioma sarcoma (v. rare)

* Treatment :-

- ① Conservative ttt : [in early cases] $\rightarrow$ Rest & foot elevation, Massage, elastic stocking, Diuretics, Antibiotics & Anti Filarial.
- ② Surgical ttt :- [in chronic cases] $\rightarrow$ Removing the thick s.c tissue for cosmetic & functional reasons e.g Sistrunk operation, • Charles's (flaying) operation OR Thompson's (swiss roll) operations.

LYMPHADENOPATHY :

* Causes of Lymphadenopathy [LN enlargement] :

- ① Inflammatory :-
- ① Acute - non specific : - septic lymphadenitis .
- infection mononucleosis.
 - ② Chronic - non specific : - ch. tonsillitis & pharyngitis
- scalp infection as pediculosis capitis.
 - = specific : - Bacterial → TB. ϕ .
- Parasitic → Filariasis
- Viral → Cat scratch, AIDS
- Protozoal → Toxoplasma.

- ② Lymphoma :-
- ① Hodgkin lymphoma.
 - ② Non-Hodgkins lymphoma.
 - ③ Burkitt's lymphoma.

- ③ Blood diseases :-
- ① Acute leukaemia
 - ② Chronic myeloid Leukemia.
 - ③ Chronic lymphatic leukemia.

- ④ others → as metastasis, collagen disease & Lipoidosis.

LYMPHOMAS

* Definition :- Primary malignant neoplasm of RES, it can be

- ↳ Hodgkin's L.
- ↳ Non Hodgkin's L.
- ↳ Burkitt's L.

I - BURKITT'S LYMPHOMA

* Definition :- The rarest type of Lymphosarcoma, it is of unknown etiology but related to EBV "Ebstein-Barr virus".

* C/P :- (Common in < 12 yrs in Western Africa), usually painless, progressive Jaw swelling is the usual presentation [may affect kidney, bone, ovary, CNS]

* Pathology :- Starry skin appearance = pale retinaculum & dark lymphocytes.

* Treatment :- chemotherapy.

	II HODGKIN'S LYMPHOMA	III NON-HODGKIN'S
incidence	- Commonst type of lymphoma.	Rare type, may associated = SLE, Sjogren's syndrome
site:	- <u>Nodal</u> ^(common) : Mainly cervical then generalized - <u>Extranodal</u> (rare): - in late stage → Liver, spleen	- <u>Nodal</u> (less common) → cervical. - <u>Extra N.</u> (More common) → abdominal (Liver, spleen, GIT mucosa,)
Pathology	- <u>N/E</u> : LN replaced by pink neoplasm w doesn't infiltrate surrounding (v. late) - <u>Micro</u> : Pleomorphism of cells + Reed-Sternberg cells : → w is pathognomic giant cell = multinuclei 2-8 in mirror image 	- <u>N/E</u> : LN replaced by white N. that infiltrate surrounding - <u>Micro</u> : - normal architecture of LN. are lost by tumour cells, lymphocyte, Retikocyte...
classific.	(Based on histological type) • Lymphocyte dominant type → Best prognosis. • Lymphocyte depleted type → worst " • Nodular sclerosing type. • Mixed cellularity type.	(Based on cell origin) • B-cell lymphoma • T-cell lymphoma • Lymphoblastic lymphoma • Histocytic lymphoma.
C/P	• young , male > Female • Rubbery Painless LN, discrete & different ^{size} • <u>Extranodal</u> → <u>systemic symptoms</u> : High sweating, wt loss, pruritis anemia, Pel-Ebstein fever → (2 wk fever alternate = 2 wk free), alcohol → ↑ pain • S/S of pressure of LN at surrounding.	• Elderly , M > F • Firm Painless LN. (may soft → if degenerative) (may hard → if calcified) • Extranodal features in case of infiltration as Hodgkin's
t/t	- Stage I (A & B), II A → Radiotherapy alone - Stage II B → Chemotherapy (MOPP) as 6 cycles supplemented by Radiotherapy - May surgery as → splenectomy (hypersplenism) → Decompression (pressure s/s)	- Radio & Chemotherapy according to stage. - May surgery to resect LN in gastric & intestinal lymphoma
prognosis	relatively good prognosis (5 yr survival 80% if proper t/t)	very bad.

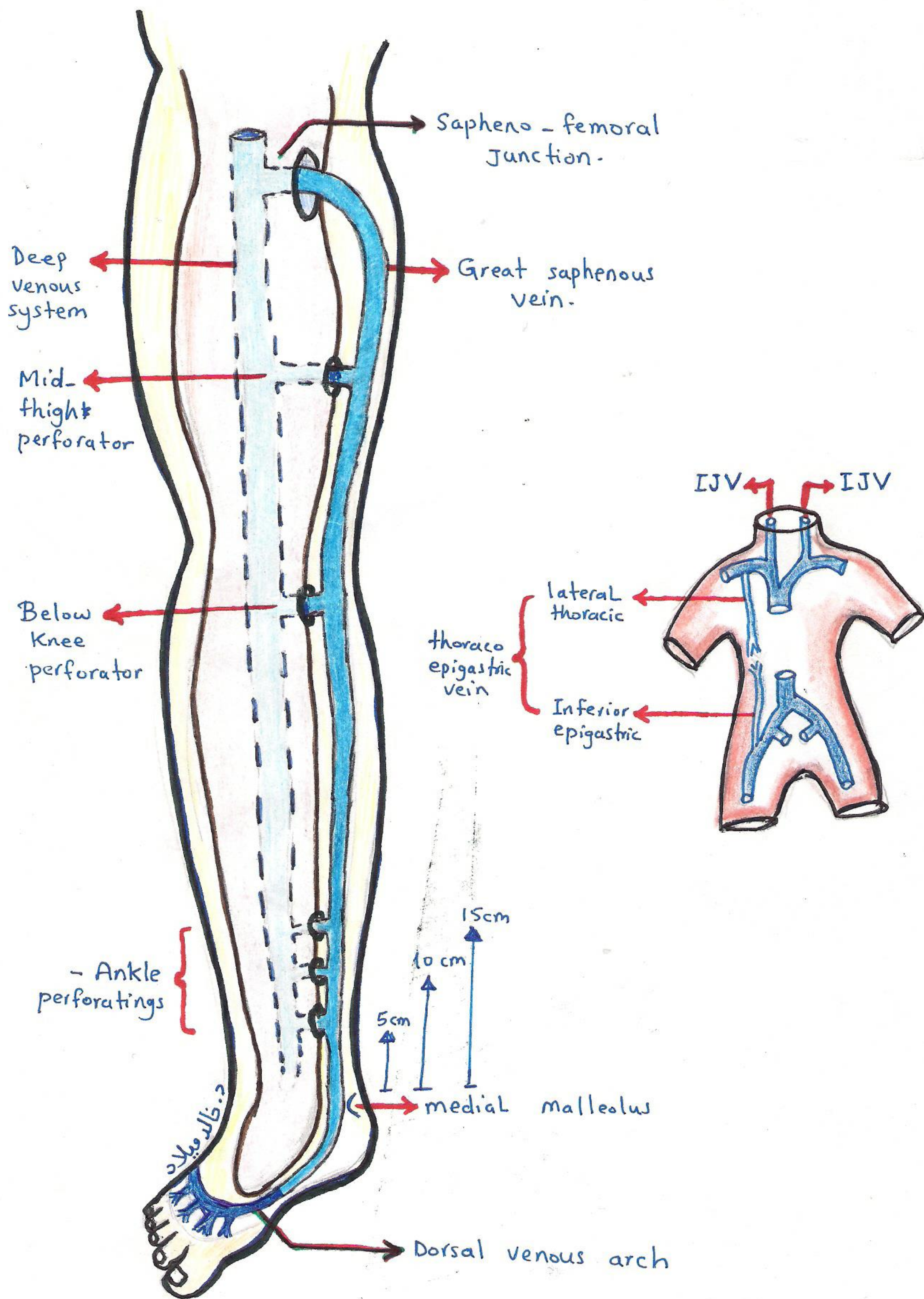
* Staging of lymphoma (Hodgkin & non-H):

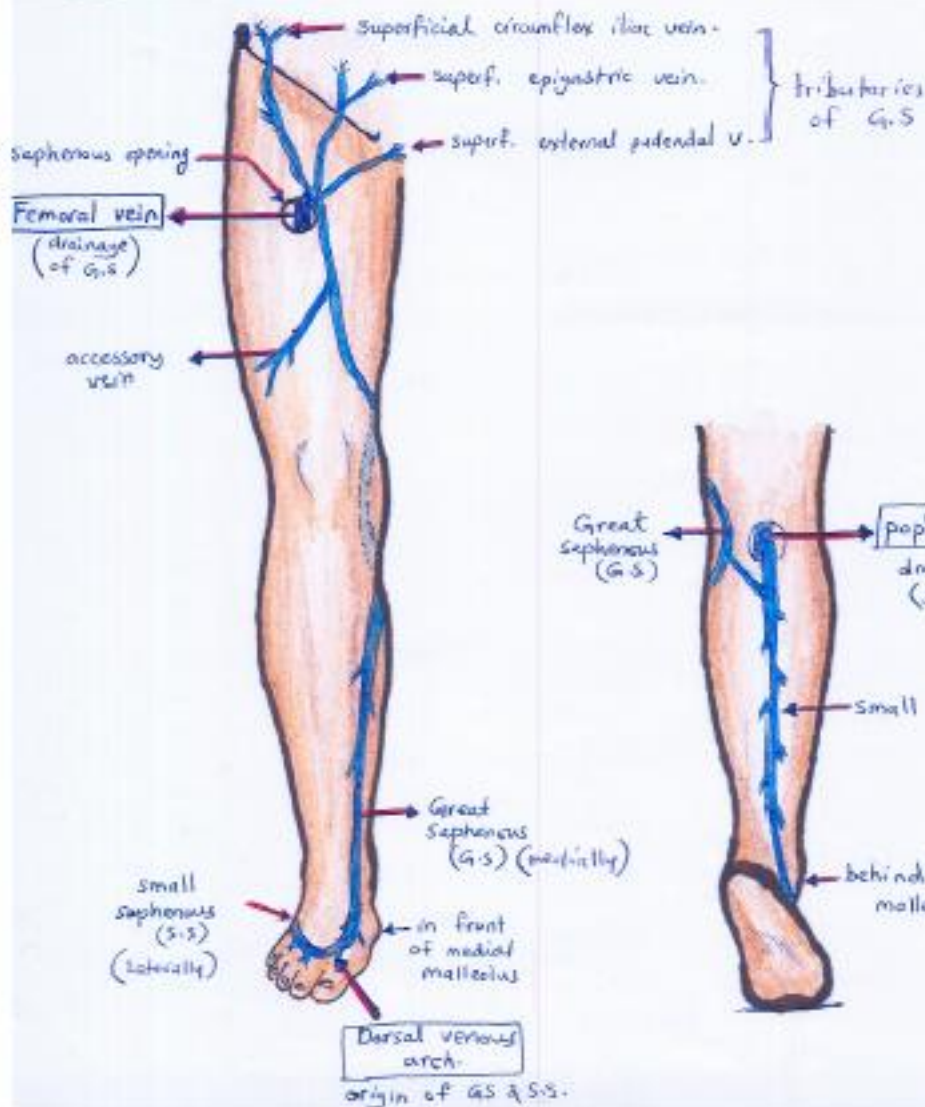
- Stage I: - one group of LNs only.
- Stage II: - More than one gp. LN same side of diaphragm.
- Stage III: - LN. involved above & below diaphragm.
- Stage IV: - Extranodal spread as liver, spleen, BM, ...

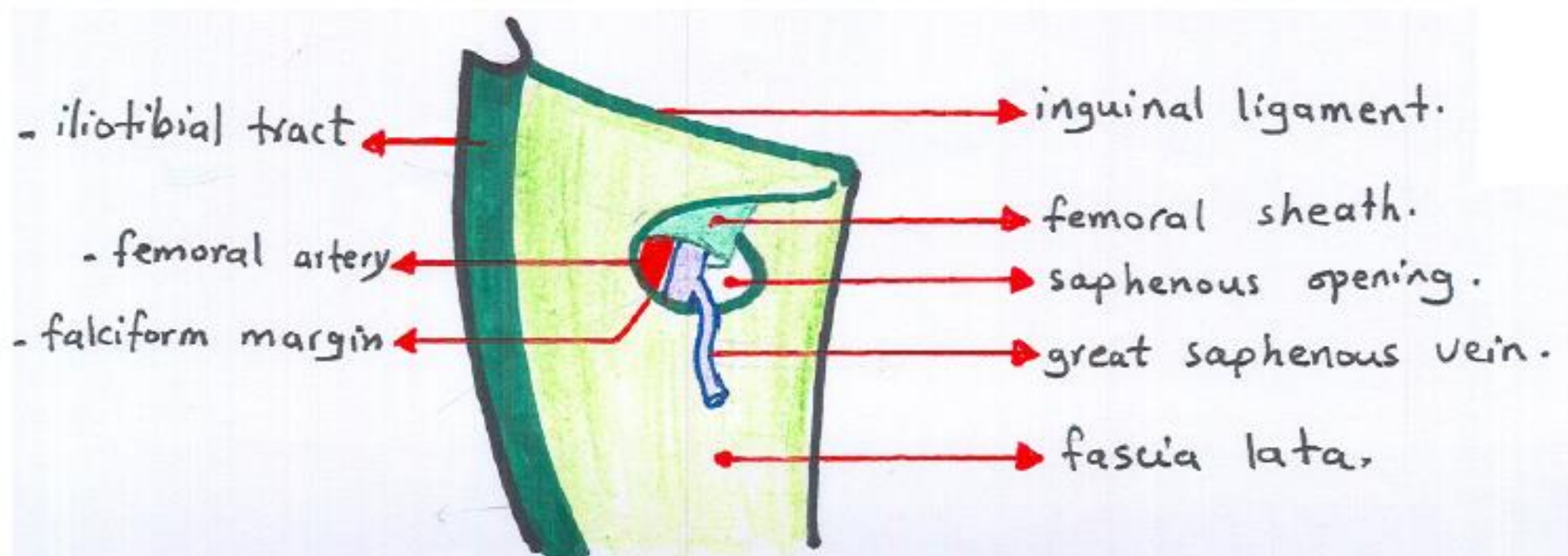
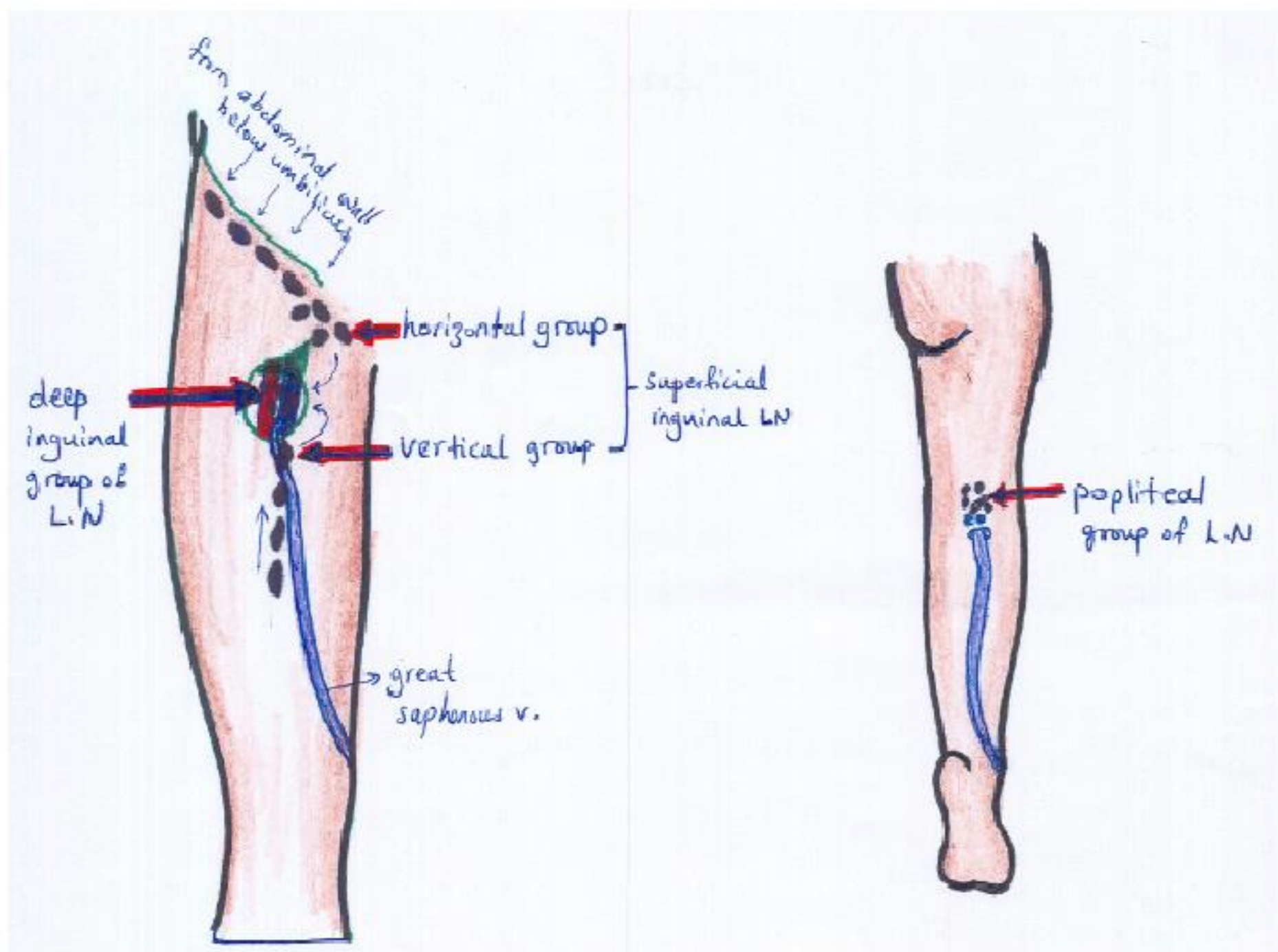
any stage may be

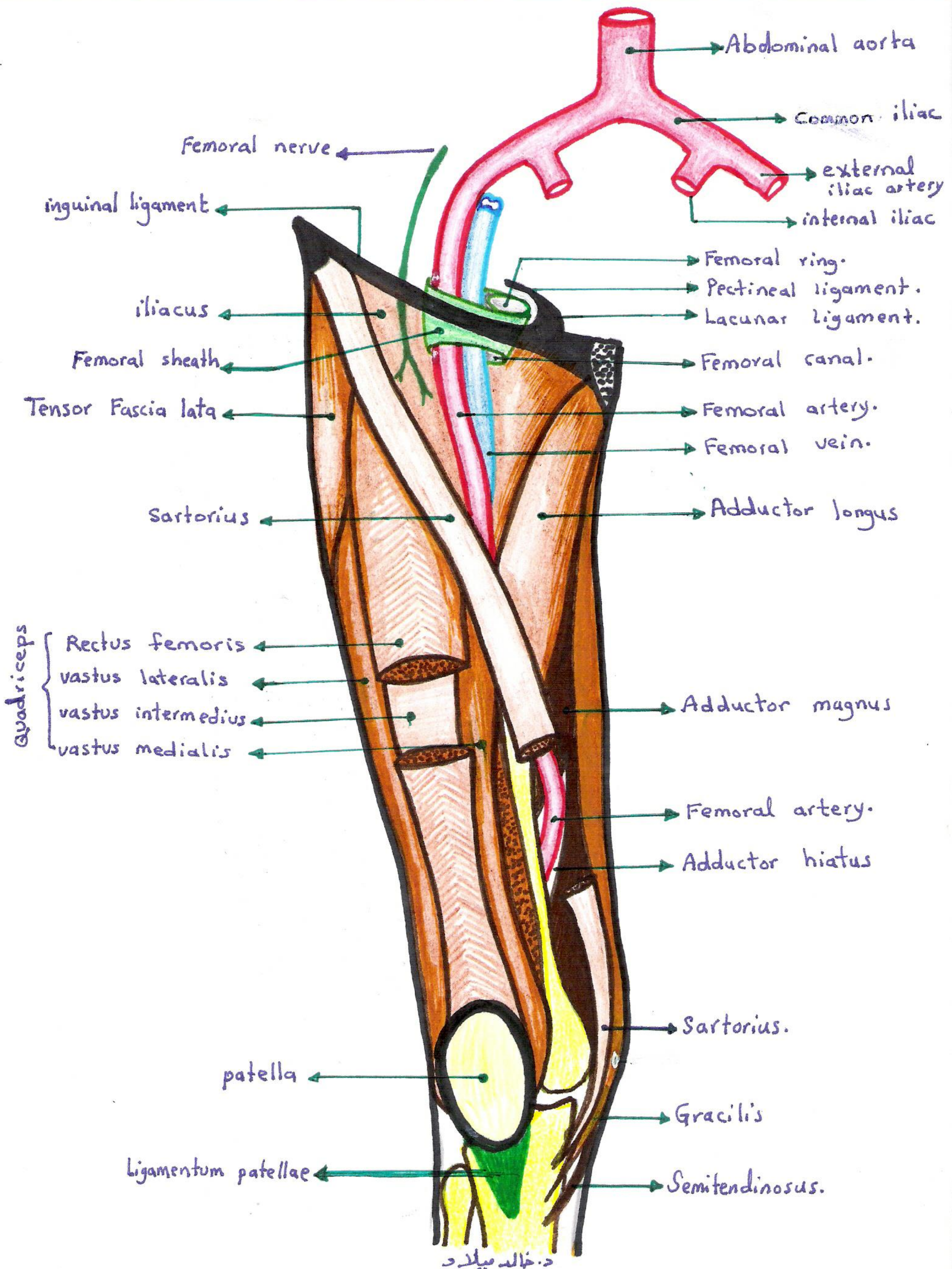
(A) → No systemic s/s

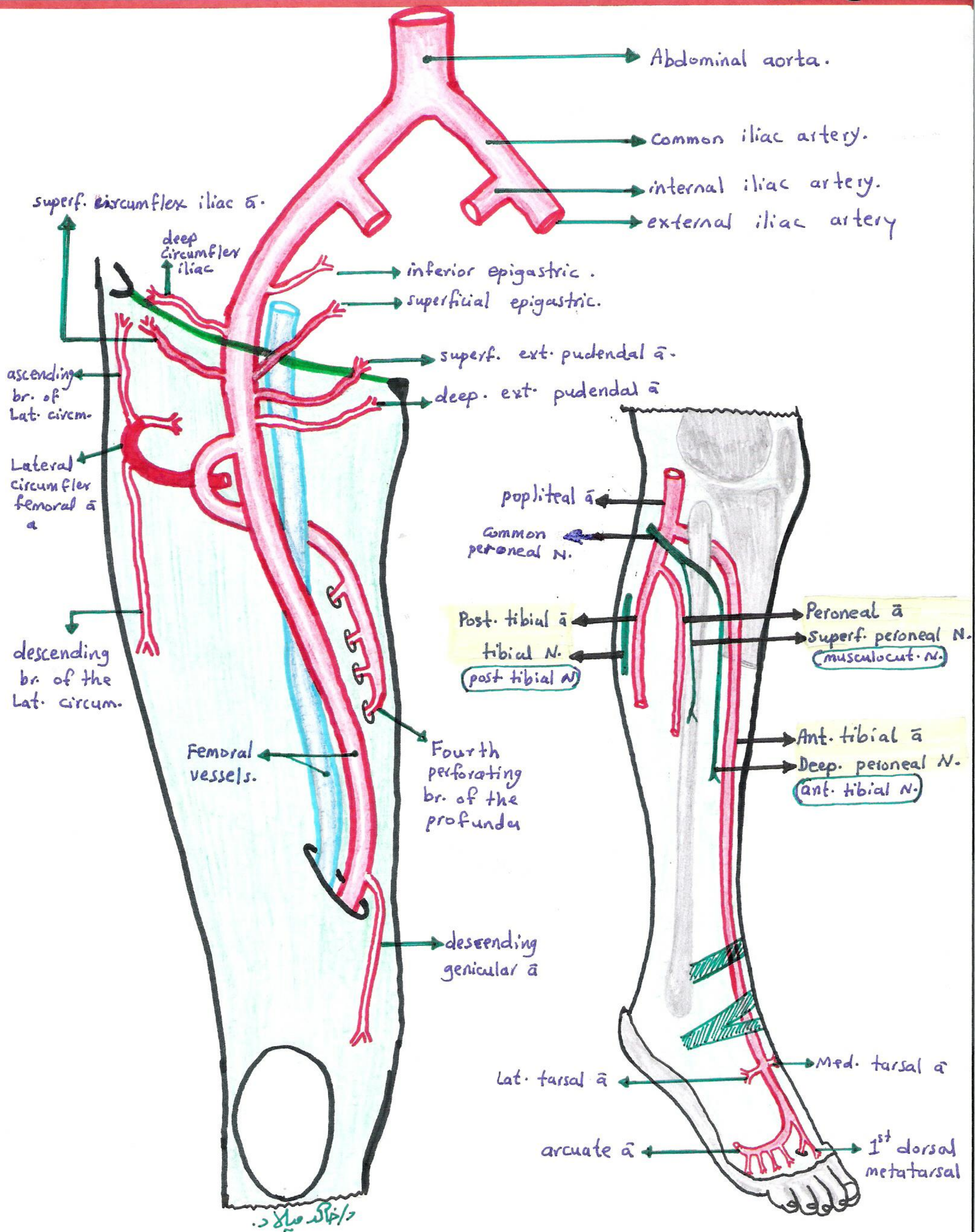
(B) → = systemic
symptoms
(one or more)















Gangrene

From Wikipedia, the free encyclopedia

For the American football team nicknamed "Gang Green," see *New York Jets*.



This article **needs additional citations for verification**.

Please help [improve this article](#) by adding [reliable references](#). Unsourced material may be [challenged](#) and [removed](#).

(February 2008)

Gangrene is a serious and potentially life-threatening condition that arises when a considerable mass of body tissue dies ([necrosis](#)).^{[1][2]} This may occur after an injury or infection, or in people suffering from any chronic health problem affecting [blood circulation](#).^[2] The prime cause of gangrene is reduced blood supply to the affected tissues, which results in [cell death](#).^[3] [Diabetes](#) and long-term smoking increase the risk of suffering from gangrene.^{[2][3]}

There are different types of gangrene with different symptoms, such as [dry gangrene](#), [wet gangrene](#), [gas gangrene](#), [internal gangrene](#) and [necrotising fasciitis](#).^{[1][2]} Treatment options include [debridement](#) (or, in severe cases, [amputation](#)) of the affected body parts, [antibiotics](#), [vascular surgery](#), [maggot therapy](#) or [hyperbaric oxygen therapy](#).^[4]

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Gangrene

Classification and external resources



Diabetic ulceration with central "dry" gangrene and, toward the edges, wet gangrene with some ascending [cellulitis](#)

ICD-10	R02. ↗ , I70.2 ↗ , E10.2 ↗ , I73.9 ↗
ICD-9	040.0 ↗ , 785.4 ↗
DiseasesDB	19273 ↗
MeSH	D005734 ↗

Etymology

[\[edit\]](#)

The [etymology](#) of gangrene derives from the [Latin](#) word "gangraena" and from the [Greek](#) gangraina (γάγγραινα), which means "putrefaction of tissues". It has no etymological connection with the word [green](#), despite the affected areas turning black and/or green and/or yellowish brown. It is coincidence that, in [Lowland Scots](#) the words "gang green" (going green) can be said to be an [eggcorn](#) for gangrene, as it describes the symptoms of the affliction.

Causes

[\[edit\]](#)

Gangrene is caused by [infection](#) or [ischemia](#), such as by the bacteria *Clostridium perfringens*^[5] or by [thrombosis](#) (blocked [blood vessel](#)). It is usually the result of critically insufficient [blood](#) supply (e.g., [peripheral vascular disease](#)) and is often associated with [diabetes](#) and long-term smoking. This condition is most common in the lower [extremities](#). The best treatment for gangrene is [revascularization](#) (i.e., restoration of blood flow) of the affected organ, which can reverse some of the effects of necrosis and allow healing. Other treatments include [debridement](#) and surgical [amputation](#). The method of treatment is, in general, determined depending on location of affected tissue and extent of tissue loss. Gangrene may appear as one effect of [foot binding](#).

Types of gangrene

[\[edit\]](#)

Dry gangrene

[\[edit\]](#)

Dry gangrene begins at the distal part of the limb due to [ischemia](#) and often occurs in the toes and feet of elderly patients due to [arteriosclerosis](#). Dry gangrene spreads slowly until it reaches the point where the blood supply is inadequate to keep tissue viable. The affected part is dry, shrunken and dark black, resembling [mummified](#) flesh. The dark coloration is due to liberation of [hemoglobin](#) from hemolyzed red blood cells, which is acted upon by [hydrogen sulfide](#) (H₂S) produced by the bacteria, resulting in formation of black iron sulfide that remains in the tissues.^[6] The line of separation usually brings about complete separation with eventual falling off of the gangrenous tissue if it is not removed surgically.

If the blood flow is interrupted for a reason other than severe bacterial infection, the result is a case of dry gangrene. People with impaired peripheral blood flow, such as diabetics, are at greater risk of developing dry gangrene.

The early signs of dry gangrene are a dull ache and sensation of coldness in the affected area along with [pallor](#) of the flesh. If caught early, the process can sometimes be reversed by vascular surgery. However, if necrosis sets in, the affected tissue must be removed just as with

The early signs of dry gangrene are a dull ache and sensation of coldness in the affected area along with [pallor](#) of the flesh. If caught early, the process can sometimes be reversed by vascular surgery. However, if necrosis sets in, the affected tissue must be removed just as with wet gangrene.

Wet gangrene

[\[edit\]](#)

Wet gangrene occurs in naturally moist tissue and organs such as the mouth, bowel, lungs, cervix, and vulva.^{[*[citation needed](#)*]} [Bedsores](#) occurring on body parts such as the sacrum, buttocks, and heels — although not necessarily moist areas — are also categorized as wet gangrene infections. In wet gangrene, the tissue is infected by saprogenic microorganisms (*Bac. perfringens*, *fusiformis*, *putrificans*, etc.), which cause tissue to swell and emit a fetid smell. Wet gangrene usually develops rapidly due to blockage of venous and/or arterial blood flow. The affected part is saturated with stagnant blood, which promotes the rapid growth of bacteria. The toxic products formed by bacteria are absorbed causing systemic manifestation of [septicemia](#) and finally death. The affected part is edematous, soft, putrid, rotten and dark. The darkness in wet gangrene occurs due to the same mechanism as in dry gangrene.

Gas gangrene

[\[edit\]](#)

Main article: [Gas gangrene](#)

Gas gangrene is a bacterial infection that produces gas within tissues. It is a deadly form of gangrene usually caused by [Clostridium perfringens](#) bacteria. Infection spreads rapidly as the gases produced by bacteria expand and infiltrate healthy tissue in the vicinity. Because of its ability to quickly spread to surrounding tissues, gas gangrene should be treated as a [medical emergency](#).

Gas gangrene is caused by a bacterial [exotoxin](#)-producing clostridial species, which are mostly found in soil and other anaerobes (e.g., [Bacteroides](#) and anaerobic [streptococci](#)). These environmental bacteria may enter the muscle through a wound and subsequently proliferate in necrotic tissue and secrete powerful toxins. These toxins destroy nearby tissue, generating gas at the same time. A gas composition of 5.9% hydrogen, 3.4% carbon dioxide, 74.5% nitrogen, and 16.1% oxygen was reported in one clinical case.^[7]

Gas gangrene can cause [necrosis](#), gas production, and [sepsis](#). Progression to [toxemia](#) and [shock](#) is often very rapid.

Other

[\[edit\]](#)

- [Noma](#) is a gangrene of the face.
- [Necrotizing fasciitis](#) affects the deeper layers of the skin.

[Fournier gangrene](#) usually affects the male genital and groin

Other

[\[edit\]](#)

- [Noma](#) is a gangrene of the face.
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- [Fournier gangrene](#) usually affects the male genitals and groin.

Treatment

[\[edit\]](#)

Treatment is usually surgical [debridement](#), with amputation necessary in many cases. Antibiotics alone are not effective because they do not penetrate infected muscles sufficiently.^{[\[citation needed\]](#)}

History

[\[edit\]](#)

As early as 1028, when antibiotics had not yet been discovered, [fly maggots](#) were commonly^{[\[citation needed\]](#)} used to treat chronic wounds or [ulcers](#) to prevent or arrest necrotic spread, as some species of [maggots](#) consume only dead flesh, leaving nearby living tissue unaffected. This practice largely died out after the introduction of [antibiotics](#), [acetonitrile](#)^{[\[citation needed\]](#)} and [enzyme](#) to the range of treatments for wounds. In recent times, however, [maggot therapy](#) has regained some credibility and is sometimes employed with great efficacy in cases of chronic tissue necrosis.

Gallery

[\[edit\]](#)



Valvless veins

1- emissary vein

2- Batson venous plexus

3- facial vein

4- vana cava

5- avernous sinus